pre-erythrocytic development of the parasites takes place in the parenchymal cells of the liver with the production of merozoites which invade the erythrocytes to establish the blood infection. At the same time strong evidence has been adduced to suggest that this developmental cycle in the liver persists after the establishment of the erythrocytic infection, may even exist without the latter, is relatively unaffected by the immunity response of the host, and is the probable source of relapses of the disease.

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Medical Memoranda

Death from Atropine Poisoning

The following rare case is interesting enough to be placed on record.

CASE REPORT

A boy aged 2 years 9 months was brought to hospital on May 20, 1950, for the treatment of an alternating convergent squint which his mother had noticed five months previously. Atropine ointment 1% was prescribed and approximately 90 gr. (6 g.) was dispensed. The mother was given a glass rod with printed instructions about the method of administration.

The ointment was first used on May 23, when two applications were given, the first in the afternoon and the second in the evening. The mother stated that the application was made by first dipping the rod into the ointment, and that the amount applied was quite small. Next morning the child was not well and the parents called in their doctor, but a further application of the ointment was given. On the morning of May 25 the child had no breakfast, and at 9.15 a.m. he had a fit (tonic and clonic contractions) and was semiconscious afterwards. At 11 a.m. he had another fit, and was admitted to All Saints' Hospital, Chatham, at 2.20 p.m. under the care of Dr. Sadiq.

On admission his temperature was 97° F. (36.1° C.), pulse 120, and respirations 32. He was a well-nourished child. He had sordes on his lips; petechiae, mainly on the left side of the trunk; dilated pupils, not reacting to light; and slight opisthotonus. The cerebrospinal fluid was normal in every way. There was no growth of organisms from a throat swab. The white blood cells numbered 28,000 per c.mm. (polymorphs 72%, lymphocytes 23%, monocytes 5%). The blood sugar at 4 p.m. on the 25th was 30 mg. per 100 ml. Next day he had petechial haemorrhages on his neck, upper chest, elbows, and forearms, and a papular rash on his legs. His blood pressure was 130/60. His voice was hoarse; he cried without shedding tears and was restless. The blood urea was 60 mg. per 100 ml. He died at 9.45 p.m.

Post-mortem Examination.—This was carried out at 3.30 p.m. on May 27 by Dr. H. G. Close. The body was that of a well-nourished male child of stated age. The eyes were sunken and the pupils dilated. Bruises were found on the tip of the right iliac crest and below the left ankle. The air passages and lungs were dry. Several petechiae were present on the anterior aspect of the heart (weight 3 oz.—85 g.). There was a very small vegetation on the mitral valve. The thymus weighed 1 oz. (28 g.), the spleen 2 oz. (57 g.), and the lungs 9 oz. (255 g.). The liver (18 oz.—510 g.) was very pale, with several petechiae on the surface; microscopically, extensive fatty changes were seen. No gross abnormality was found in the kidneys (3 oz.—85 g.). The brain (44 oz.—1.25 kg.) was mushy, soft, and tore easily, and petechiae were present in the corpus callosum. The clinical story and post-mortem appearances are consistent with the diagnosis of atropine poisoning.

COMMENT

An analysis of the ointment showed that it contained 0.8% of atropine alkaloid, and the analyst stated that at least seven-eighths of the original quantity of the ointment dispensed was sent to him for analysis. Approximately ½ gr. (32 mg.) of ointment was picked up on the glass rod at each application. The total quantity administered, therefore, was approximately 3 gr. (0.2 g.), containing 1.6 mg. of alkaloid. The possibility that the child may have swallowed some of the ointment was considered, but almost all the ointment originally dispensed was accounted for.

Morton (1939) reported nine cases of atropine poisoning, two of which were fatal. In the first case six drops of 1% atropine solution were dropped into an injured eye, and after operation 1.5 g. of 1% atropine ointment was placed on the dressing next to the eye. Death occurred fourteen hours later. The total amount of atropine administered was 18.1 mg. In the second fatal case the pupils of both eyes were dilated with 1% atropine solution. Death occurred in under twenty-four hours and the total amount of atropine administered was 2 mg.

Hughes (1938) recorded a case, not fatal, in which 1% atropine ointment was smeared into both eyes twice daily for three days. On the third day the child was reported to be ill with a temperature, and the skin was flushed and very dry to the touch. Morphine and pilocarpine were given and recovery was uneventful.

My thanks are due to Dr. M. Sadiq for permission to publish this case and to Dr. H. G. Close for the pathological report.

W. E. HEATH, M.B., B.S., D.O.

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Neurological Complication After Combined Diphtheria and Pertussis Immunization

The occurrence of severe complications after an injection of pertussis vaccine is rare enough to merit record.

CASE REPORT

A girl aged 1 year was admitted to hospital on July 17, 1948, with left hemiplegia. She had been a healthy infant. There was no family or personal history of convulsions or allergy. Injections of diphtheria A.P.T. on May 9 and 19 had caused no apparent untoward effect. An injection of pertussis vaccine on June 17 similarly had caused no ill effect. On July 15 a mixture of alum-precipitated pertussis vaccine and diphtheria A.P.T. had been injected, and a few hours later she is said to have been quieter than usual, but by bedtime (about 17½ hours later) had become her usual self again. Early next morning she had been found in a fit with twitching of the face and both limbs on the left side, and 2½ gr. (0.16 g.) of phenobarbitone had been given. Shortly afterwards a state of semi-coma had ensued, lasting several hours, and later a left hemiplegia had developed.

On admission the child was very irritable, her temperature was 102° F. (38.9° C.). Physically she was well nourished. She had a left hemiplegia with little movement of the upper limb and none of the lower. The reflexes of the upper limb were absent, the knee-jerks were present and equal, but the left ankle-jerk was absent. The right plantar response was flexor but the left was indefinite. The abdominal reflexes were absent. There was no detectable involvement of the cranial nerves and the fundi oculorum were normal. The pyrexia subsided in two days.

On January 17 the spinal fluid was under normal pressure. It contained no excess of cells; 30 mg. of protein per 100 ml.; no excess of globulin; 730 mg. of chlorides per 100 ml. On January 20 a repeat tap showed that the fluid contained no excess of cells; 30 mg. of protein per 100 ml.; no excess of globulin; 720 mg. of chlorides per 100 ml.; 65 mg. of sugar. No organisms were grown. Queckenstedt's test was negative. The Wassermann reaction of the blood and spinal fluid was negative.

The hemiplegia improved rapidly, all the deep reflexes of the limbs and abdominal reflexes appearing within a week. The patient was discharged on July 31, a happy and lively baby with slight weakness of the affected limbs.

When seen again on August 5 there was no apparent weakness of the limbs. Her mother had noticed some clumsiness of the affected limbs in play, but considered that this was improving. There was no evidence of mental deficiency.

COMMENT

Anderson and Morris (1950) have reviewed the literature and reported a case which developed convulsions 36 hours after a first injection of combined diphtheria–pertussis antigen. Their case had had a previous history of convulsions.

The present case is of interest because no apparent ill-effects had been produced by previous injections of A.P.T. and alum-precipitated pertussis vaccine given singly; the convulsion followed the injection of a mixture of these two antigens, made by the same manufacturer. Sensitization as a result of the previous injections cannot be ruled out, but the frequency of allergic states and the rarity of serious neurological complications of this kind suggest that these reactions may have a genetic basis; a previous history of convulsions may therefore, pending further additions to our knowledge, be regarded as a contraindication to the use of pertussis antigens. It should not be forgotten that an attack of whooping-cough may cause encephalopathy with permanent residua, including mental changes (Levy and Perry, 1948).

J. VAHRMAN, M.B., B.S., C.P.H.,
Senior Registrar, Department of Infectious Diseases, Royal Free Hospital.

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An Emergency Leucotomy

Prefrontal leucotomy is now an established procedure in psychiatry. The indications for such an operation are usually regarded as certain forms of chronic mental illness which have not responded to more conservative methods. In the following case the operation may be correctly described as life-saving; and it would not be inaccurate to speak here of "an emergency leucotomy."

CASE REPORT

The patient, a woman aged 21, was admitted to hospital on August 6, 1947, with a provisional diagnosis of anorexia nervosa. She was the youngest child of a family of eight, whose father had committed suicide at the age of 54. Her childhood and adolescence had passed without revealing any abnormal personality traits. At 19 she became sensitive about her appearance and fastidious about her food, and developed the delusion that she was unable to swallow and that she might become "filled up with wind" and die. She would pretend to chew her food but secretly spit it out and hide it in odd corners. Her sleep was disturbed and she was losing weight. On admission her weight was 5 st. (31.8 kg.); two years previously it had been 7 st. (44.5 kg.). Apart from this peculiarity about eating, there were no other psychotic symptoms.

A course of modified insulin was given and strict hospital routine and observation were maintained. Her weight gradually returned to normal and she was discharged home on December 13 "happy, sociable, and eating and sleeping well."

After remaining well at home for four months her previous delusions and habits returned, and she was readmitted to hospital on June 30, 1948, in a dangerous state of emaciation. Her urine was loaded with acetone and her appearance was that of a concentration-camp victim. Her weight was 5 st. 5 lb. (34 kg.). She refused all food and required tube-feeding. Later, when her physical health was stronger, a course of E.C.T. was given. This treatment produced only very temporary remission, and within a few days of stopping it the refusal of food began again. Tube-feeding had to be resorted to. This was a painful procedure for all concerned, as the patient did all in her power to resist and to regurgitate the food. She made frequent promises to eat, but was a genius at deception. Food would be found hidden in the strangest places. She showed no insight into the danger she was in and could explain her refusal to eat only by saying "she came of a thin family."

A transorbital leucotomy was performed on December 6, 1948. This produced a temporary remission, but by March 13, 1949, she had returned to her previous state and tube-feeding had again to be resorted to.

It was at this stage that the emergency occurred. She developed a recurrent evening pyrexia. A radiograph of her chest showed "a cavity below the right clavicle with a little surrounding opacity, doubtless tuberculous." Her erythrocyte sedimentation rate at this date was 37 mm. in 1 hour. Further E.C.T. was now out of the question, and the prognosis was indeed grave. Her emaciated state and continued dependence on tube-feeding pointed to a fulminating phthisis soon ending the story.

A full leucotomy seemed now the only possible hope. This was performed on May 4. Convalescence was uneventful and she ate her food satisfactorily from that day forward. Before operation her weight was 5 st. 6 lb. (34.5 kg.) and a week after it was 5 st. 12 lb. (37.2 kg.). On June 27 it was 8 st. (50.8 kg.) and her erythrocyte sedimentation rate was 7 mm. in 1 hour.

In spite of the presence of a tuberculous cavity it was decided not to confine her to bed. The after-treatment of a leucotomized patient should be one of continued occupation, and this was considered a matter of priority in her case. Her activity was gradually increased and no rise of temperature was noticed. As the weeks passed her mental state improved concurrently with her physical condition. She became more interested in other people and in her own appearance, and her work at the occupational therapy class improved. On June 9 a further *x*-ray report read: "Cavity cleared below right clavicle and infection almost gone."

COMMENT

This case seems worthy of record for several reasons. (1) It raises an interesting point in psychiatric diagnosis. The initial symptoms were classically those of an anorexia nervosa, but the long duration of the illness, the temporary response to E.C.T., and the more permanent response to leucotomy suggest that basically the illness was schizophrenic. (2) It shows that when a transorbital leucotomy has proved a failure a full prefrontal operation may yet be a success. (3) The influence of the mental state on a patient suffering from phthisis is well known. In the above case a specifically psychiatric procedure and the ignoring of the physical disease during convalescence led to an eventual healing of a proved tubercular lesion—a healing as rapid as any collapse treatment would have produced.

My thanks are due to Dr. J. N. P. Moore, medical superintendent of St. Patrick's Hospital, Dublin, and to Professor A. McConnell and Mr. J. P. Lanigan, of the Neurosurgical Unit, St. Laurence's Hospital, Dublin, for their assistance with this case.

M. O'C. DRURY, M.D., D.P.M.,
Deputy Medical Superintendent, St. Patrick's Hospital, Dublin.